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      Set  Items  Description
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Cost is in DialUnits
? b 410
       10dec08 13:15:54 User208760 Session D3004.1
            $0.56    0.156 DialUnits File1
     $0.56  Estimated cost File1
     $0.56  Estimated cost this search
     $0.56  Estimated total session cost   0.156 DialUnits

File 410:Dialog Comm.-of-Interest Newsletters 2008 /Mar
       (c) 2008 Dialog

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HILIGHT set on as ''
HILIGHT set on as ''
? begin 5,73,155,399
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     $0.00  Estimated cost File410
     $0.02  TELNET
     $0.02  Estimated cost this search
     $0.58  Estimated total session cost   0.273 DialUnits

SYSTEM:OS  - DIALOG OneSearch
  File   5:Biosis Previews(R) 1926-2008/Nov W5
         (c) 2008 The Thomson Corporation
  File  73:EMBASE 1974-2008/Dec 10
         (c) 2008 Elsevier B.V.
  File 155:MEDLINE(R) 1950-2008/Dec 08
         (c) format only 2008 Dialog
  File 399:CA SEARCH(R) 1967-2008/UD=14924
         (c) 2008 American Chemical Society
*File 399: Use is subject to the terms of your user/customer agreement.
IPCR/8 classification codes now searchable as IC=. See HELP NEWSIPCR.

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? s (pemphigus) and (animal)(10n)(model?)
           18574  PEMPHIGUS
         5335125  ANIMAL
         5276040  MODEL?
         1055958  ANIMAL(10N)MODEL?
      S1     273  (PEMPHIGUS) AND (ANIMAL)(10N)(MODEL?)
? s s1 and mrl
            273  S1
          10329  MRL
      S2       0  S1 AND MRL
? s s1 and mrl?
            273  S1
          11362  MRL?
      S3       0  S1 AND MRL?
? s (pemphigus) and (animal)(10n)(model?)(20n)(correl? or predict? or lack or
difficult?)
Processing
           18574  PEMPHIGUS
         5335125  ANIMAL
         5276040  MODEL?
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         2822545  CORREL?
         1750068  PREDICT?
          653565  LACK
          740796  DIFFICULT?
           29882  ANIMAL(10N)MODEL?(20N)(((CORREL? OR PREDICT?) OR LACK) OR
                  DIFFICULT?)
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                  PREDICT? OR LACK OR DIFFICULT?)
? rd s4
      S5       6  RD S4  (unique items)
? t s5/7/all

 5/7/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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18734937   BIOSIS NO.: 200600080332
Annals of the New York Academy of Sciences
BOOK TITLE: Annals of the New York Academy of Sciences
AUTHOR: Gershwin ME; Shoeenfeld Y
BOOK AUTHOR/EDITOR: Gershwin ME (Editor); Shoeenfeld Y (Editor)
SERIES TITLE: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES  1050 2005
BOOK PUBLISHER: NEW YORK ACAD SCIENCES, 2 EAST 63RD ST, NEW YORK, NY 10021
                USA
ISSN: 0077-8923_(print)  ISBN: 1-57331-516-8 (H)
DOCUMENT TYPE: Book
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: This 431-page book is volume 1050 of the Annals of the New York
  Academy of Sciences and the focus of this volume is autoimmunity with
  respect to concepts and diagnosis. This volume comprises part of the
  proceedings of the Fourth International Congress on Autoimmunity, which
  was supported by the American Autoimmune Related Diseases Association
  (AARDA) and the Associazione Patologie Autoimmuni Internazionale (APAI)
  and was held in Budapest, Hungary, in November 2004. The volume is
  structured into 3 parts and contains 45 papers divided among these 3
  parts. Part I focuses on the principles of autoimmunity and there are 13
  papers in this first part. The focus of part II is diagnostic
  consideration of autoimmunity and there are 26 papers in this section.
  There are 4 papers in the third and final part of the book, which
  concentrates on infection and autoimmunity. In this volume both novel and
  scientific and clinical aspects of autoimmunity and autoimmune diseases
  are presented in papers written by the world's leading autoimmunologists.
  Topics covered include etiology and pathogenesis, genetics, mechanisms,
  tolerance, diagnostics, the role of hormones, infection, animal
  models, individual autoimmune diseases, therapy and the art and
  science of   ***predicting***   autoimmunity. The book is indexed by
  contributor. This book will be of interest to clinical immunologists,
  clinicians, and researchers in immunology.


 5/7/2     (Item 1 from file: 73)
DIALOG(R)File  73:EMBASE
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0081996687     EMBASE No: 2007431044
  WHO task group on environmental health criteria on principles and methods
for assessing autoimmunity associated with exposure to chemicals
  ISSUE TITLE: Principles and Methods for Assessing Autoimmunity Associated
with Exposure to Chemicals
```

Chauhan R.S.; Cohen Tervaert J.W.; Conrad K.; Cooper G.S.; De Souza Querioz M.L.; Germolec D.R.; Hall A.J.; Ohsawa M.; Philen R.M.; Pieters R.H.H.; Rose N.R.; Van Loveren H.; Vos J.G.; Vickers C.; Damoiseaux J.; De Jong W.H.; Van Londen K.; Colosio C.; Corsini E.; Descotes J.; Lovik M.; Luster M.I.; Pallardy M.; Kunz S.
Department of Pathology, College of Veterinary Sciences, G.B. Pant University of Agriculture and Technology, Pantnagar, India; Centre for Animal Disease Research and Diagnosis, Indian Veterinary Research Institute, Izatnagar, India
CORRESP. AUTHOR/AFFIL: Chauhan R.S.: Department of Pathology, College of Veterinary Sciences, G.B. Pant University of Agriculture and Technology, Pantnagar, India

Environmental Health Criteria (Environ. Health Criteria) (Switzerland) December 1, 2006, -/236 (xi-333)
CODEN: EHCRD ISSN: 0250-863X ISBN: 9241572361 ISBN: 9789241572361
DOCUMENT TYPE: Book Series; Conference Paper RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English; French; Spanish
NUMBER OF REFERENCES: 960

Autoimmunity is characterized by the reaction of cells (autoreactive T lymphocytes) or products (autoantibodies) of the immune system against the organism's own antigens (autoantigens). It may be part of the physiological immune response ("natural autoimmunity") or pathologically induced, which may eventually lead to development of clinical abnormalities ("autoimmune diseases"). Many different autoimmune diseases can occur, but all are characterized by the inappropriate or excessive immune response against autoantigens, leading to chronic inflammation, tissue destruction, and/or dysfunction. To date, more than 60 diseases have a proven or strongly suspected autoimmune etiology. Generally, autoimmune diseases are perceived to be relatively uncommon. However, when all autoimmune diseases are combined, the estimated prevalence is high (3-5% of the general population), which underlines their importance to public health. Because of difficulties in diagnosis and in designing and standardizing epidemiological studies, limited data are available, and the prevalence may actually be underestimated. Nonetheless, there is epidemiological evidence of increasing prevalence of certain autoimmune diseases in highly industrialized countries, which cannot be attributed to better diagnosis alone. Furthermore, there is growing evidence that autoimmune mechanisms may play a role in many other diseases (atherosclerosis, for instance). Autoimmune diseases are multifactorial. Both intrinsic factors (e.g. genetics, hormones, age) and environmental factors (e.g. infections, diet, drugs, environmental chemicals) may contribute to the induction, development, and progression of autoimmune diseases. Environmental factors are believed to be a major factor responsible for their increased prevalence. Environmental factors operating in a genetically susceptible host may directly initiate, facilitate, or exacerbate the pathological immune process, induce mutations in genes coding for immunoregulatory factors, or modify immune tolerance or regulatory and immune effector pathways. Drug-induced autoimmune or autoimmune-like disorders and hypersensitivity are of major concern and often the reason for withdrawing drugs from the market or restricting their use. Systemic allergy is not well understood and is often considered idiosyncratic, but it may be of an allergic or autoimmune nature. We have learned much about the mechanisms of idiosyncratic autoimmune diseases by studying the autoimmune phenomena that result from exposure to therapeutics. In addition, there have been several "point source" outbreaks of autoimmune diseases due to environmental exposures to chemicals such as Spanish toxic oil and L-tryptophan that have advanced our knowledge substantially. There is now considerable epidemiological evidence pertaining to the association between occupational exposure to crystalline silica dust (quartz) and the risk of several

systemic autoimmune diseases (specifically, systemic sclerosis, systemic lupus erythematosus, rheumatoid arthritis, and systemic small vessel vasculitis). Epidemiological studies also support a role of occupational exposure to solvents in the development of systemic sclerosis, but a clear consensus has not developed on the specific exposures or classes of chemicals involved and whether this association extends to other diseases. Some autoimmune diseases (e.g. Graves disease, rheumatoid arthritis) have been associated with tobacco use, particularly among current smokers, but only weak or no associations have been seen with other diseases. Additional experimental research examining the effects of these and other chemical and physical agents, using exposure routes relevant to the human experience in occupational settings or in environmental contamination, is needed to advance our understanding of the pathogenesis of autoimmune diseases. In contrast to the available studies concerning silica, solvents, and smoking, there are relatively few epidemiological data pertaining to the effect of dioxins, pesticides, or heavy metals on the development or progression of autoimmune diseases. There is also some research on the influence of dietary factors on autoimmune diseases. This is a broad area that includes caloric intake, specific nutrients and foods, and dietary supplements. Coeliac disease is an example of an autoimmune disease with a clear dietary link in which an immunological response to specific proteins in wheat, barley, and rye produces autoantibodies directed against tissue transglutaminase, causing mucosal damage in the small intestine. It is highly likely that infection plays a role in many autoimmune disorders, although the infectious agent and mechanism by which it causes disease may differ from one disorder to another. Most hypotheses relating infection to autoimmunity have assumed that infection plays a direct causal role, although it may simply serve as a predisposing factor. Infectious agents may play a role due to sequence homology with endogenous proteins, resulting in "molecular mimicry", and also may act as "priming" agents due to non-specific/polyclonal stimulation of immune factors such as cytokines and co-stimulatory molecules. Hygienic status, resulting in a lack of infectious stimuli, may have an impact on autoimmunity. Chemical agents may play an important role in interacting with infections, an area that has been poorly studied. There exist a variety of methods to detect enhanced antibody formation and autoantibodies in humans and experimental animals following environmental exposure. In contrast, tests available for measuring the potential of chemicals or environmental factors to produce autoimmune disease or augment existing autoimmune disease are not readily available. A large number of animal models exist that have been used primarily to explore basic mechanisms and therapeutic possibilities for certain autoimmune diseases. Etiology in the various models is based on genetic predisposition, induction with specific antigens (mostly in combination with an adjuvant), or challenge with infectious agents. Chemical-induced autoimmune disease models are less common. In addition, autoimmunogenic and allergenic effects of compounds are usually not identified in routine toxicity studies, in part because outbred animals are used and relevant parameters are not studied. In addition, outliers are usually discarded from the experiment, whereas in fact outliers may indicate unexpected and idiosyncratic immune effects. A general strategy to assess the autoimmunogenic potential of chemicals is lacking. One promising approach is the popliteal lymph node assay. This represents a straightforward and robust animal test model that may be used to link direct lymphocyte node reactions to local application of potentially immunoactive chemicals. However, these assays may be predictive of the sensitizing potential, but not necessarily of the autoimmunogenic potential, of agents and do not represent a systemic route of exposure. The burden on health and heavy costs of autoimmune diseases highlight their importance with regard to risk assessment. Risk assessment of autoimmunity associated with chemical or physical agents should consider available epidemiological data, hazard identification and dose-response

data derived from animal and human studies, data related to mode of action, and susceptibility factors. The risk assessment process may eventually help to calculate the cost of autoimmune disease associated with exposure to chemical and physical agents. Currently, the risk assessment for agents that are suspected of inducing or exacerbating autoimmunity or autoimmune diseases is hampered by the fact that appropriate information is not available, particularly validated animal models. Because of the individual- and population-level burden of autoimmune disease, risk assessment with respect to this group of diseases assumes special importance.

5/7/3 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

0081593739 EMBASE No: 2007027026
Toxic epidermal necrolysis
Pereira F.A.; Mudgil A.V.; Rosmarin D.M.
Departments of Dermatology, Mount Sinai School of Medicine; New York Medical College
AUTHOR EMAIL: carozat@aol.com
CORRESP. AUTHOR/AFFIL: Pereira F.A.: Departments of Dermatology, Mount Sinai School of Medicine
CORRESP. AUTHOR EMAIL: carozat@aol.com

Journal of the American Academy of Dermatology (J. Am. Acad. Dermatol.) (United States) February 1, 2007, 56/2 (181-200)
CODEN: JAADD ISSN: 0190-9622
PUBLISHER ITEM IDENTIFIER: S0190962206012230
DOI: 10.1016/j.jaad.2006.04.048
DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 216

Toxic epidermal necrolysis (TEN) is an unpredictable, life-threatening drug reaction associated with a 30% mortality. Massive keratinocyte apoptosis is the hallmark of TEN. Cytotoxic T lymphocytes appear to be the main effector cells and there is experimental evidence for involvement of both the Fas-Fas ligand and perforin/granzyme pathways. Optimal treatment for these patients remains to be clarified. Discontinuation of the offending drug and prompt referral to a burn unit are generally agreed upon steps. Beyond that, however, considerable controversy exists. Evidence both pro and con exists for the use of IVIG, systemic corticosteroid, and other measures. There is also evidence suggesting that combination therapies may be of value. All the clinical data, however, is anecdotal or based on observational or retrospective studies. Definitive answers are not yet available. Given the rarity of TEN and the large number of patients required for a study to be statistically meaningful, placebo controlled trials are logistically ***difficult*** to accomplish. The absence of an ***animal*** ***model*** further hampers research into this condition. This article reviews recent data concerning clinical presentation, pathogenesis and treatment of TEN. Learning objectives: At the conclusion of this learning activity, participants should have acquired a more comprehensive knowledge of our current understanding of the classification, clinical presentation, etiology, pathophysiology, prognosis, and treatment of TEN.

5/7/4 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

0080979595 EMBASE No: 2006039547
Role of animal models in the study of drug-induced hypersensitivity reactions
Uetrecht J.
Department of Pharmacology, Clinical Pharmacology and Toxicology, University of Toronto, Toronto, Ont., Canada
AUTHOR EMAIL: jack.uetrecht@utoronto.ca
CORRESP. AUTHOR/AFFIL: Uetrecht J.: Department of Pharmacology, Clinical Pharmacology and Toxicology, University of Toronto, Toronto, Ont., Canada
CORRESP. AUTHOR EMAIL: jack.uetrecht@utoronto.ca

AAPS Journal (AAPS Journal) (United States) January 13, 2006, 7/4 (12)
CODEN: PHARF ISSN: 1522-1059 eISSN: 1522-1059
DOI: 10.1208/aapsj070489
URL: http://www.aapspharmsci.org/view.asp?art=aapsj070489
ARTICLE NUMBER: 89
DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 50

Drug-induced hypersensitivity reactions (DHRs) are a major problem, in large part because of their unpredictable nature. If we understood the mechanisms of these reactions better, they might be ***predictable*** . Their unpredictable nature also makes mechanistic studies very ***difficult*** , especially prospective clinical studies. ***Animal*** models are vital to most biomedical research, and they are almost the only way to test basic hypotheses of DHRs, such as the involvement of reactive metabolites. However, useful ***animal*** ***models*** of DHRs are rare because DHRs are also unpredictable in animals. For example. sulfonamide-induced DHRs in large-breed dogs appear to be valid because they are very similar to the DHRs that occur in humans, however. the incidence is only (similar)0.25%, and large-breed dogs are ***difficult*** to use as an ***animal*** ***model*** . Two more practical ***models*** are
penicillamine-induced autoimmunity in the Brown Norway rat and nevirapine-induced skin rash in rats, The toxicity in these models is clearly immune mediated. In other models, such as amodiaquine-induced agranulocytosis/hepatotoxicity and halothane-induced hepatotoxicity, the drug induces an immune response but there is no clinical toxicity. This finding suggests that regulatory mechanisms usually limit toxicity. Many of the basic characteristics of the penicillamine and nevirapine models, such as memory and tolerance, are quite different suggesting that the mechanisms are also significantly different. More animal models are needed to study the range of mechanisms involved in DHRs; without them, progress in understanding such reactions is likely to be slow.

5/7/5 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

0078286678 EMBASE No: 2000336270
New aspects in the pathogenesis of bullous pemphigoid
Neue aspekte zur pathogenese des bullosen pemphigoids
Schmidt E.; Brocker E.-B.; Zillikens D.
Universitats-Hautklinik, Wurzburg, Germany
CORRESP. AUTHOR/AFFIL: Zillikens D.: Universitats-Hautklinik, Josef-Schneider-Strasse 2, 97080 Wurzburg, Germany
CORRESP. AUTHOR EMAIL: zillikens-d.derma@mail.uni-wuerzburg.de

Hautarzt (Hautarzt) (Germany) October 4, 2000, 51/9 (637-645)
CODEN: HAUTA ISSN: 0017-8470
DOI: 10.1007/s001050051188
DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract
LANGUAGE: German SUMMARY LANGUAGE: English; German
NUMBER OF REFERENCES: 82

Bullous pemphigoid (BP) is a subepidermal blistering autoimmune disease of the elderly. Autoantibodies are directed against two hemidesmosomal proteins, designated BP180 and BP230. While BP230 localizes intracellularly and associates with the hemidesmosomal plaque, BP180 is a transmembrane glycoprotein with an extracellular domain consisting of approximately 1000 amino acids. The non-collagenous (NC) 16A domain, that encompasses 76 amino acids and localizes directly adjacent to the transmembrane region, has been identified as an immunodominant region of the BP180 ectodomain. In the majority of BP sera, circulating antibodies to BP180 NC16A are detected; their serum levels correlate with disease activity. Neonatal mice that are injected with rabbit anti-murine BP180 antibodies develop a BP-like subepidermal blistering disease demonstrating the biological importance of antibodies to BP180. The pathogenically relevant site on murine BP180 corresponds to a stretch of the NC16A domain on human BP180. In contrast to pemphigus, in BP, autoantibodies alone are not sufficient to induce blisters. In addition, complement activation, the infiltration of inflammatory cells and the release of proteases and various inflammatory mediators, including cytokines, are essential for lesion formation. In this review, we give an up-date on the pathogenesis of BP.

5/7/6 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

0075723043 EMBASE No: 1994152442
Bullous pemphigoid sera induce bullous-pemphigoid-like lesions in neonatal mice pretreated with a limited dose of ultraviolet B irradiation
Mitsuhashi Y.; Nakano H.; Murai T.; Ohta T.; Sawamura D.; Hanada K.; Hashimoto I.
Department of Dermatology, Hirosaki University School Medicine, 5 Zaifu-cho, Hirosaki 036, Japan
CORRESP. AUTHOR/AFFIL: Mitsuhashi Y.: Department of Dermatology, Hirosaki University School Medicine, 5 Zaifu-cho, Hirosaki 036, Japan

Dermatology (DERMATOLOGY) (Switzerland) May 25, 1994, 189/SUPPL. 1 (76-81)
CODEN: DERAE ISSN: 1018-8665
DOCUMENT TYPE: Journal; Conference Paper RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English

The role of bullous pemphigoid (BP) autoantibodies (Abs) in the pathogenesis of BP is unclear. ***Lack*** of a confirmed experimental animal model prevents studies of the pathogenetic role of BP Absolute We hypothesized that some alterations of BP antigens (BP Ags) will be necessary for causative binding of BP Abs to the Ags. To cause an artificial and reproducible alteration, neonatal mice were irradiated with ultraviolet B (UVB). After the irradiation of 600 mJ/cm SUP 2 of UVB, BP serum was intraperitoneally injected. When the BP sera, which recognized only the 230-kD BP Ag, or both 230- and 180-kD BP Ags, were transferred into the UVB-treated mice, erosions and vesicles appeared in 14-78% of the animals. Histopathological examination revealed subepidermal blister formation in the mice treated with UVB and BP sera. Electron microscopy

demonstrated that the separation occurred at the level of the lamina lucida. Human IgG and C3 were deposited at the dermal-epidermal junction. Control animals to which healthy sera were injected after the same dose of UVB irradiation showed degeneration of the upper epidermis but no apparent dermal-epidermal cleft formation histopathologically. These results suggest that BP antibodies play a pathogenetic role in vivo. This animal model can contribute to a study of the pathogenesis of BP.

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? e au=amagai masayuki ?

Ref   Items  Index-term
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E2      225  AU=AMAGAI MASAYUKI
E3        0 *AU=AMAGAI MASAYUKI ?
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E6        8  AU=AMAGAI N
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E9        4  AU=AMAGAI NAOMI
E10       3  AU=AMAGAI NORIKO
E11       1  AU=AMAGAI R
E12       5  AU=AMAGAI R.

          Enter P or PAGE for more
? s e1-e3
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            225  AU=AMAGAI MASAYUKI
              0  AU=AMAGAI MASAYUKI ?
      S6    226  E1-E3
? s s6 and pemphigus
            226  S6
          18574  PEMPHIGUS
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? rd s7
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           8893  CD40L
            908  MR1
          36212  CD40
         560821  LIGAND
          17004  CD40(W)LIGAND
           3938  CD154
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? t s9/3/all

 9/3/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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19097585   BIOSIS NO.: 200600442980
Tolerance induction by the blockade of CD40/CD154 interaction in
  pemphigus vulgaris mouse model
AUTHOR: Aoki-Ota Miyo; Kinoshita Mari; Ota Takayuki; Tsunoda Kazuyuki;
  Iwasaki Toshiro; Tanaka Sigeru; Koyasu Shigeo; Nishikawa Takeji;
  Amagai Masayuki (Reprint)
AUTHOR ADDRESS: Keio Univ, Sch Med, Dept Dermatol, Shinjuku Ku, 35
  Shinanomachi, Tokyo 1608582, Japan**Japan
AUTHOR E-MAIL ADDRESS: amagai@sc.itc.keio.ac.jp
JOURNAL: Journal of Investigative Dermatology  126 (1): p105-113 JAN 2006
2006
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ISSN: 0022-202X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
? t s9/7/all

 9/7/1   (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)

19097585   BIOSIS NO.: 200600442980
Tolerance induction by the blockade of CD40/CD154 interaction in
  pemphigus vulgaris mouse model
AUTHOR: Aoki-Ota Miyo; Kinoshita Mari; Ota Takayuki; Tsunoda Kazuyuki;
  Iwasaki Toshiro; Tanaka Sigeru; Koyasu Shigeo; Nishikawa Takeji;
  Amagai Masayuki (Reprint)
AUTHOR ADDRESS: Keio Univ, Sch Med, Dept Dermatol, Shinjuku Ku, 35
  Shinanomachi, Tokyo 1608582, Japan**Japan
AUTHOR E-MAIL ADDRESS: amagai@sc.itc.keio.ac.jp
JOURNAL: Journal of Investigative Dermatology 126 (1): p105-113 JAN 2006
2006
ISSN: 0022-202X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Pemphigus vulgaris (PV) is an autoimmune blistering disease
  caused by IgG autoantibodies against desmoglein 3 (Dsg3). We have
  recently developed an active disease mouse model for PV by adoptive
  transfer of splenocytes from Dsg3(-/-) mice. The purpose of this study
  was to determine the role of CD40/CD154 interaction in the
  pathogenic antibody production and development of the disease in PV model
  mice. When anti- ***CD154***   monoclonal antibody (mAb) was administered to
  recipient mice prior to adoptive transfer, anti-CD154 mAb almost
  completely blocked the anti- Dsg3 IgG production and prevented blister
  formation. The blockade of CD40/ ***CD154***   interaction induced tolerance
  against Dsg3 as the suppression of antibody production was observed
  through day 70, and it was maintained even after challenge by
  immunization with recombinant mouse Dsg3 or by adoptive transfer of
  immunized Dsg3(-/-) splenocytes. Furthermore, the tolerance to Dsg3 was
  transferable because cotransfer of splenocytes from anti-CD154
  mAb-treated mice and naive Dsg3(-/-) splenocytes significantly suppressed
  anti- Dsg3 IgG production in recipient mice. In contrast, when anti-
  CD154 mAb was injected after the mice had developed the PV
  phenotype, no significant suppression of the production of anti- Dsg3 IgG
  was observed. These findings indicate that the CD40/ ***CD154***
  interaction is essential for the induction of pathogenic anti- Dsg3 IgG
  antibodies and that antigen-specific immune-regulatory cells induced by
  anti-CD154 mAb would hold a therapeutic option for autoimmune
  diseases.
? ds

Set	Items	Description
S1	273	(PEMPHIGUS) AND (ANIMAL)(10N)(MODEL?)
S2	0	S1 AND MRL
S3	0	S1 AND MRL?
S4	6	(PEMPHIGUS) AND (ANIMAL)(10N)(MODEL?)(20N)(CORREL? OR PREDICT? OR LACK OR DIFFICULT?)
S5	6	RD S4 (unique items)
S6	226	E1-E3
S7	158	S6 AND PEMPHIGUS

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     S11       6  RD S10  (unique items)
? t s11/3/all

 11/3/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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16047103   BIOSIS NO.: 200100218942
Viewing cell-cell adhesion through a lens of pemphigus
AUTHOR: Amagai Masayuki (Reprint)
AUTHOR ADDRESS: Department of Dermatology, Keio University School of
  Medicine, Tokyo, Japan**Japan
JOURNAL: Cell Structure and Function  25 (6): p373 December, 2000
2000
MEDIUM: print
CONFERENCE/MEETING: Fifty-third Annual Meeting of the Japan Society for
Cell Biology  Fukuoka, Japan  October 31-November 02, 2000; 20001031
SPONSOR: Japan Society for Cell Biology
ISSN: 0386-7196
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English


 11/3/2     (Item 2 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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15829162   BIOSIS NO.: 200100001001
Use of domain-swapped molecules for conformational epitope mapping of
  desmoglein 3 in Pemphigus vulgaris
AUTHOR: Futei Yuko; Amagai Masayuki (Reprint); Sekiguchi Maiko;
  Nishifuji Koji; Fujii Yoshiko; Nishikawa Takeji
AUTHOR ADDRESS: Department of Dermatology, Keio University School of
  Medicine, 35 Shinanomachi, Shinjuku-ku, Tokyo, 160-8582, Japan**Japan
JOURNAL: Journal of Investigative Dermatology  115 (5): p829-834 November,
2000 2000
MEDIUM: print
ISSN: 0022-202X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English


 11/3/3     (Item 3 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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15777848   BIOSIS NO.: 200000496161
Thymoma with pemphigus foliaceus
AUTHOR: Takeshita Kei (Reprint); Amano Maki; Shimizu Takayuki; Oyamada
  Yoshitaka; Abiko Tomohiro; Kobayashi Koichi; Futei Yuko; Amagai
  Masayuki; Kuramochi Shigeru; Asano Koichiro; Yamaguchi Kazuhiro
AUTHOR ADDRESS: Department of Medicine, Keio University School of Medicine,
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35 Shinanomachi, Shinjuku-ku, Tokyo, 160-8582, Japan**Japan
JOURNAL: Internal Medicine (Tokyo) 39 (9): p742-747 September, 2000
2000
MEDIUM: print
ISSN: 0918-2918
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

11/3/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15658915 BIOSIS NO.: 200000377228
Use of autoantigen-knockout mice in developing an active autoimmune disease model for pemphigus
AUTHOR: Amagai Masayuki (Reprint); Tsunoda Kazuyuki; Suzuki Harumi; Nishifuji Koji; Koyasu Shigeo; Nishikawa Takeji
AUTHOR ADDRESS: Department of Dermatology, Keio University School of Medicine, 35 Shinanomachi, Shinjuku-ku, Tokyo, 160-8582, Japan**Japan
JOURNAL: Journal of Clinical Investigation 105 (5): p625-631 March, 2000
2000
MEDIUM: print
ISSN: 0021-9738
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

11/3/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15618119 BIOSIS NO.: 200000336432
Brief report: Protection against pemphigus foliaceus by desmoglein 3 in neonates
AUTHOR: Wu Hong; Wang Zhi Hong; Yan Albert; Lyle Stephen; Fakharzadeh Steven; Wahl James K; Wheelock Margaret J; Ishikawa Hiroyasu; Uitto Jouni ; Amagai Masayuki; Stanley John R (Reprint)
AUTHOR ADDRESS: Department of Dermatology, University of Pennsylvania School of Medicine, 415 Curie Blvd., 211B Clinical Research Bldg., Philadelphia, PA, 19104-6142, USA**USA
JOURNAL: New England Journal of Medicine 343 (1): p31-35 July, 2000
2000
MEDIUM: print
ISSN: 0028-4793
DOCUMENT TYPE: Article
RECORD TYPE: Citation
LANGUAGE: English

11/3/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15498736 BIOSIS NO.: 200000217049
Detection of antigen-specific B cells in patients with pemphigus vulgaris by enzyme-linked immunospot assay: Requirement of T cell collaboration for autoantibody production
AUTHOR: Nishifuji Koji; Amagai Masayuki (Reprint); Kuwana Masataka;

Iwasaki Toshiro; Nishikawa Takeji
AUTHOR ADDRESS: Department of Dermatology, Keio University School of Medicine, 35 Shinanomachi, Shinjuku-ku, Tokyo, 160-8582, Japan**Japan
JOURNAL: Journal of Investigative Dermatology 114 (1): p88-94 Jan., 2000
2000
MEDIUM: print
ISSN: 0022-202X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
? t s11/7/4

11/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15658915 BIOSIS NO.: 200000377228
Use of autoantigen-knockout mice in developing an active autoimmune disease model for pemphigus
AUTHOR: Amagai Masayuki (Reprint); Tsunoda Kazuyuki; Suzuki Harumi; Nishifuji Koji; Koyasu Shigeo; Nishikawa Takeji
AUTHOR ADDRESS: Department of Dermatology, Keio University School of Medicine, 35 Shinanomachi, Shinjuku-ku, Tokyo, 160-8582, Japan**Japan
JOURNAL: Journal of Clinical Investigation 105 (5): p625-631 March, 2000
2000
MEDIUM: print
ISSN: 0021-9738
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The development of experimental models of active autoimmune diseases can be difficult due to tolerance of autoantigens, but knockout mice, which fail to acquire tolerance to the defective gene product, provide a useful tool for this purpose. Using knockout mice lacking desmoglein 3 (Dsg3), the target antigen of pemphigus vulgaris (PV), we have generated an active disease model for this autoantibody-mediated disease. Dsg3-/- mice, but not Dsg3+/-littermates, produced anti-Dsg3 IgG that binds native Dsg3, when immunized with recombinant mouse Dsg3. Splenocytes from the immunized Dsg3-/- mice were then adoptively transferred into Rag-2-/- immunodeficient mice expressing Dsg3. Anti-Dsg3 IgG was stably produced in the recipient mice for more than 6 months without further boosting. This IgG bound to Dsg3 in vivo and disrupted the cell-cell adhesion of keratinocytes. Consequently, the recipient mice developed erosions in their oral mucous membranes with typical histologic findings of PV. In addition, the recipient mice showed telogen hair loss, as found in Dsg3-/- mice. Collectively, the recipient mice developed the phenotype of PV due to the pathogenic anti-Dsg3 IgG. This model will be valuable for developing novel therapeutic strategies. Furthermore, our approach can be applied broadly for the development of various autoimmune disease models.
? s (pemphigus) and (animal)(10n)(model?)(20n)(autoantigen?)(20n)knockout)
>>>Unmatched parentheses
? s (pemphigus) and (animal)(10n)(model?)(20n)(autoantigen?)(20n)(knockout)
18574 PEMPHIGUS
5335125 ANIMAL
5276040 MODEL?
40886 AUTOANTIGEN?
136290 KNOCKOUT
43 ANIMAL(10N)MODEL?(20N)AUTOANTIGEN?(20N)KNOCKOUT
S12 3 (PEMPHIGUS) AND

(ANIMAL)(10N)(MODEL?)(20N)(AUTOANTIGEN?)(20N)(KNOCKOUT)
? rd s12
S13 3 RD S12 (unique items)
? t s13/7/all

13/7/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15931011 PMID: 15327541
A mouse model of pemphigus vulgaris by adoptive transfer of naive splenocytes from desmoglein 3 knockout mice.
Aoki-Ota M; Tsunoda K; Ota T; Iwasaki T; Koyasu S; Amagai M; Nishikawa T
Department of Dermatology, Keio University School of Medicine, 35 Shinanomachi, Shinjuku-ku, Tokyo 160-8582, Japan.
British journal of dermatology (England) Aug 2004, 151 (2) p346-54, ISSN 0007-0963--Print Journal Code: 0004041
Publishing Model Print
Document type: Journal Article; Research Support, Non-U.S. Gov't
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
BACKGROUND: Pemphigus vulgaris (PV) is an autoimmune blistering disease caused by antidesmoglein3 (anti-Dsg3) IgG autoantibodies. Recently, we developed a PV mouse model by adoptive transfer of splenocytes from recombinant Dsg3-immunized Dsg3(-/-) mice to Rag2(-/-) immunodeficient mice that expressed Dsg3. OBJECTIVES: We determined whether the adoptive transfer of naive splenocytes from nonimmunized Dsg3(-/-) mice induces the anti-Dsg3 IgG production and the PV phenoytpe in recipient mice. METHODS: We adoptively transferred naive Dsg3(-/-) splenocytes into Rag2(-/-) mice and compared their PV phenoytpe with those mice receiving immunized Dsg3(-/-) splenocytes. The numbers of splenocytes and their subpopulations required for anti-Dsg3 IgG production were examined. RESULTS: Mice that received naive Dsg3(-/-) splenocytes produced anti-Dsg3 IgG, which bound to keratinocyte cell surfaces in vivo, and developed the PV phenotype, including oral erosions with suprabasilar acantholysis. Antibody production and the appearance of the PV phenotype were delayed by approximately 2 weeks in mice that received naive splenocytes compared with mice that received immunized splenocytes. However, once the PV phenotypes developed, there were no apparent differences in disease severity between the two models. Interestingly, the anti-Dsg3 IgG titres were significantly lower in mice that received naive splenocytes than in mice that received immunized splenocytes, suggesting that the former antibodies were more potent than the latter. The frequency of anti-Dsg3 IgG production depended on the number of transferred naive splenocytes. Both CD4+ T cells and B220+ B cells from naive Dsg3(-/-) mice were essential for the production of anti-Dsg3 IgG antibodies. CONCLUSIONS: Dsg3-specific naive lymphocytes in Dsg3(-/-) mice can be primed and activated by the endogenous Dsg3 in recipient mice to produce pathogenic anti-Dsg3 IgG without active immunization. This approach using naive lymphocytes provides a unique model to dissect immunological mechanisms of tolerance against peripheral autoimmune targets.
Record Date Created: 20040825
Record Date Completed: 20041216

13/7/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15049849 PMID: 12574390

Induction of pemphigus phenotype by a mouse monoclonal antibody against the amino-terminal adhesive interface of desmoglein 3.

Tsunoda Kazuyuki; Ota Takayuki; Aoki Miyo; Yamada Taketo; Nagai Tetsuo; Nakagawa Taneaki; Koyasu Shigeo; Nishikawa Takeji; Amagai Masayuki
Department of Dermatology, Keio University School of Medicine, 35 Shinanomachi, Shinjuku-ku, Tokyo 160-8582, Japan.

Journal of immunology (Baltimore, Md. - 1950) (United States) Feb 15 2003, 170 (4) p2170-8, ISSN 0022-1767--Print Journal Code: 2985117R
Publishing Model Print
Document type: Journal Article; Research Support, Non-U.S. Gov't
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

Pemphigus vulgaris (PV) is a life-threatening autoimmune blistering disease that is caused by IgG autoantibodies against the cadherin-type adhesion molecule desmoglein (Dsg)3. Previously, we have generated an active mouse model for PV by adoptive transfer of Dsg3(-/-) splenocytes. In this study, we isolated eight AK series, anti-Dsg3 IgG mAbs from the PV mouse model, and examined their pathogenic activities in induction of blister formation. Intraperitoneal inoculation of the AK23 hybridoma, but not the other AK hybridomas, induced the virtually identical phenotype to that of PV model mice or Dsg3(-/-) mice with typical histology of PV. Epitope mapping with domain-swapped and point-mutated Dsg1/Dsg3 molecules revealed that AK23 recognized a calcium-dependent conformational epitope on Dsg3, which consisted of the V3, K7, P8, and D59 Dsg3-specific residues that formed the adhesive interface between juxtaposed Dsg, as predicted by the crystal structure. The epitopes of the mAbs that failed to show apparent pathogenic activity were mapped in the middle to carboxyl-terminal extracellular region of Dsg3, where no direct intermolecular interaction was predicted. These findings demonstrate the pathogenic heterogeneity among anti-Dsg3 IgG Abs due to their epitopes, and suggest the direct inhibition of adhesive interaction of Dsg as an initial molecular event of blister formation in ***pemphigus*** .

Record Date Created: 20030207
Record Date Completed: 20030425

13/7/3 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

137092745 CA: 137(7)92745v PATENT
Use of autoantigen-knockout mice in developing an active autoimmune disease model for pemphigus
INVENTOR(AUTHOR): Amagai, Masayuki; Nishikawa, Takeji; Koyasu, Shigeo
LOCATION: Japan,
ASSIGNEE: Keio University
PATENT: Japan Tokkyo Koho ; JP 3306625 B1 DATE: 20020724
APPLICATION: JP 2001156126 (20010524)
PAGES: 7 pp. CODEN: JTXXFF LANGUAGE: Japanese
PATENT CLASSIFICATIONS:
CLASS: A01K-067/027A; C12N-015/00B
SECTION:
CA215008 Immunochemistry
CA203XXX Biochemical Genetics
CA209XXX Biochemical Methods
CA214XXX Mammalian Pathological Biochemistry
IDENTIFIERS: desmoglein 3 mouse model pemphigus vulgaris
DESCRIPTORS:
Antigens...
autoantigens, gene knockout mice; use of autoantigen-knockout mice in

developing active autoimmune disease model for pemphigus
Glycoproteins...
desmoglein, 3 (Dsg3), autoantigen, gene knockout mice; use of autoantigen-knockout mice in developing active autoimmune disease model for pemphigus
Skin,disease...
pemphigus vulgaris; use of autoantigen-knockout mice in developing active autoimmune disease model for pemphigus
Proteins...
RAG-2 (recombination-activating gene, 2), gene knockout mice; use of autoantigen-knockout mice in developing active autoimmune disease model for pemphigus
Gene,animal...
RAG2, knockout mice; use of autoantigen-knockout mice in developing active autoimmune disease model for pemphigus
Spleen...
splenocyte, transplantation of; use of autoantigen-knockout mice in developing active autoimmune disease model for pemphigus
Transplant and Transplantation...
splenocyte; use of autoantigen-knockout mice in developing active autoimmune disease model for pemphigus
Animal... Autoimmune disease... Disease models... Gene targeting... Mouse ...
use of autoantigen-knockout mice in developing active autoimmune disease model for pemphigus
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